White mice which had received injection of 0.5 to 2.0 grm. invariably died in from 12 to 28 hours, with symptoms of acute ascending paralysis. The nerve cells, especially those of the anterior horns of the spinal cord, showed acute degeneration, with chromatolysis, alteration in position of the nucleus, vacuolization, etc. In the brain hyperemia and small hemorrhages were found, but the peripheral nerves and muscles were unaltered. Guinea pigs proved quite susceptible, and after the injection into the peritoneal cavity of 10.0-15.0 grm., died in from 1 to 5 days, showing progressive emaciation and weakness. Rabbits died in convulsions 4 to 6 hours after the intravenous injection of 10.0 grm. or more of the toxine.

In both rabbits and guinea pigs it was found that a condition of chronic intoxication could be produced by repeated injections of less than the lethal dose. In both animals paresis or paralysis of the hind legs usually came on in from 1 month to 6 weeks. His conclusions are:

In acute intoxication the changes consisted in alteration (chromatolysis, excentric position of the nucleus and vacuolization) of the nerve cells, especially of those of the anterior horns, to a less extent of those of the intervertebral ganglia, least of those of the nuclei of the medulla and of the cortex.

In chronic intoxication, degenerative neuritis was most common, and sometimes degeneration of the posterior roots and of the posterior columns of the cord was observed. ALLEN.

40. STUDIEN ZUR PATHOLOGIE UND PATHOLOGISCHEN ANATOMIE DER RÜCKENMARKSCOMPRESSION BEI WIRBELCARIES (Studies on the Pathology and Pathological Anatomy of Compression of the Spinal Cord in Vertebral Caries). Alfred Fickler (Deutsche Zeitschrift für Nervenheilkunde, 16, 1899, p. 1).

Fickler has had a very rich material at his disposal for a study of vertebral caries—20 cases in all, 19 of which were with necropsy. Some of his more important statements are: Tuberculosis of the spine is almost always secondary, and usually the lungs are the primary seat of infection, although the lymph glands are occasionally first affected, and more rarely other organs. Trauma seems to be of some importance in the development of the disease, although a cause is not usually found. The disease seems to appear somewhat more frequently after middle life, as in more than 50 per cent. of Fickler's cases the patients were over 50 years old. The first symptom is usually dull pain at the affected vertebræ, and is increased by bending over, but in children the pain is not infrequently absent, and in adults also it may be unimportant. Rigidity of the spine is another early symptom. Pain on pressure over the spine is absent in about half of the cases. Root symptoms, usually sensory, are among the earliest symptoms of the disease, and the pain is continuous, although it may occur periodically. The first symptoms of compression of the spinal cord are disturbances of motor innervation, such as fatigue, ataxia, and paresis. Paraplegia may occur suddenly, and when it does it is due to rupture of the abscess into the vertebral canal or to the giving away of carious vertebræ. One of the earliest symptoms of compression of the spinal cord is constipation. Unfortunately, Fickler has had no experience in operation in cases of Pott's disease. The most common cause of compression of the spinal cord is the extension of the tuberculous process to the epidural tissue.

A great many histological details are given by Fickler, among which the most interesting is the description of fibers within the cord, believed by him to be newly-formed nerve fibers. From this finding he concludes that the nerve fibers of the spinal cord are capable of regeneration with complete restoration of function, provided the vascular system is intact. The recovery of function after spinal compression, he believes, depends upon the restoration of the normal lymph circulation, upon the formation of medullary sheaths about persisting axis cylinders, and upon the regeneration of nerve fibers. SPILLER.

41. ZUR PATHOLOGISCHEN ANATOMIE UND BACTERIOLOGIE DES DELIRIUM ACUTUM (The Pathological Anatomy and Bacteriology of Acute Delirium). A. D. Kazowsky (Centralbl. f. allg. Path. u. path. Anat., 10, 1899, p. 489).

After a brief summary of the important literature upon the subject, particularly that of Russian authors, two cases are reported in which it was possible to make extensive histological investigations, and in one of them also bacteriological studies. The first patient, a man, was brought to the hospital in a state of intense excitement, with destructive tendencies, and fever. There were hallucinations of sight, hyperesthesia, refusal of food, and occasionally vomiting. The patient did not sleep, became weak, and died on the sixth day of his residence in the hospital. At the autopsy, aside from hyperemia of the central nervous system and of the viscera, there was a slight tumor found upon the anterior vermiform process of the cerebellum. Microscopically, there was found distention of the blood vessels, peri-vascular and cellular accumulation, various alterations in the nerve cells and the nerve fibers, proliferation of the neuroglia, distended spaces around the ganglion cells in which cellular elements were found; the nerve cells showed alteration of the nuclei, and disappearance or dislocation of the nucleoli. In addition many of the cells contained a considerable quantity of yellowish pigment that resisted all reagents and stained only with osmic acid. Similar collections of pigment were found in the blood vessels. This pigmentary infiltration of the ganglion cells cannot be looked upon as abnormal, excepting in the areas in which there was some softening, where the cells contained considerably more pigment than in the other situations. No cultures were made from the tissue, but microscopically micrococci occasionally arranged in chains were discovered. The second patient, a soldier, was brought to the hospital in a state of extreme depression. He soon became excited, destructive, had fever, developed swelling in both parotid glands, had extreme exhaustion, and died. The whole period of the acute process lasted fifteen days. At the autopsy there was hyperemia of the brain, enlarged spleen, cloudy swelling and perhaps necrosis of the liver, and also some punctiform hemorrhages in the mucous membranes. Cultures from the cerebrospinal fluid, the heart, blood and the spleen showed the presence of staphylococcus pyogenes aureus. Microscopically the features were similar to those in the other case. Pathologically, these two cases are identical; clinically, one represents an idiopathic acute delirium, the other an acute exacerbation of a pre-existing psychosis. Both are probably due to infection with pyogenic cocci. It appears from the study of these cases as well as from a consideration of the literature that we are not justified in considering acute delirium as an independent disease. It rather represents an acute infection, particularly localized in the central nervous system. It is difficult to explain, however, the route by which the micro-organisms reach the brain, but probably there is an infection of mild form that becomes first generalized and then localized in a brain already weakened by some other cause. However, it is also possible that the idiopathic form of acute delirium may be